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# WITHOUT CONSCIENCE OR WITHOUT ACTIVE CONSCIENCE? THE ETIOLOGY OF PSYCHOPATHY REVISITED

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ABSTRACT. Despite an impressive body of research spanning seven decades, the causes of psychopathy and psychopathic violence remain enigmatic for mental health professionals and society as a whole. A keystone of the disorder is the absence of normal human emotional experience. In recent years, a predominant view has been that a genetic predisposition is essential to its formation while environmental factors determine the course of the disorder. The present paper proposes an alternate, less common pathway to psychopathy in which environmental factors are critical ("secondary psychopathy"). Clinical and empirical evidence is reviewed supporting the hypothesis that negative childhood experiences can profoundly affect emotional functioning in adulthood. Specifically, certain individuals who are severely traumatized or disillusioned by loved ones might over time learn to "turn off" their emotions as an effective coping mechanism, later emerging as psychopathic personality disorder. It is argued that, with continued validation of the hypothesis, secondary psychopathy should be considered a distinctive dissociative disorder based on this detachment of emotion and cognition/behavior.

We only become what we are by the radical and deep-seeded refusal of that which others have made us. (Jean-Paul Sartre)

REFERENCES TO INDIVIDUALS clearly fitting the bill for prototypical psychopathy (the current construct) have surfaced since antiquity (Cleckley, 1976; Hare, 1996). Despite this long-standing recognition of psychopathic characters and the damage they incur, "the etiology of the predatory, cold-blooded nature of psychopathy remains obscure" (Hare, 1996, p. 25). The basis for a central feature of the construct — lack of empathy and, hence, conscience (or "lovelessness and guiltlessness"; McCord & McCord, 1964) — represents an enigma that continues to baffle mental health professionals and the public. Clinicians have speculated that underpinning psychopathy is an impairment or inability to experience the emotional aspects

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of life events (Williamson, Harpur, & Hare, 1991). Still, the question remains: What factors merge to create this quality and ultimately the "intraspecies predators who use charm, manipulation, intimidation, and violence to control others and to satisfy their own selfish needs" (Hare, 1996, p. 26)?

A salient feature emerging in the recent literature on psychopathy is a virtual disregard for the possible contribution of childhood experiences to the origination of the disorder. This is largely due to the failure to identify consistent negative occurrences in the backgrounds of psychopaths, despite decades of research and clinical observations. However, the present paper asserts that to wholly ignore the influence of environmental generative factors is myopic and counterproductive if one considers that psychopathy might not be a unitary disorder etiologically.

Cleckley (1982), although clearly favoring a genetic or neurological bent, suggests that in some cases experiential factors likely play a significant contributory role: "Perhaps there are . . . [some] psychopaths in whom such [psychogenic] influences play an important part" (p. 253). Cleckley later summarizes the foundation for the present paper:

In a few of the cases reported here an impressive account was given of incidents and reported reactions (with indications of emotion) that could theoretically be said to explain emotional withdrawal (despite maintenance of excellent rational contact) from the areas or levels of living in which severe hurt, deep joy, genuine pride, shame, dignity, and love are encountered and experienced. Protest reactions, loss of insight, or acting out of unconscious impulses . . . or a diatribe against life and its subjective emptiness could be interpreted as major causal factors in the disorder we encounter clinically. (p. 257)

In this paper, the argument will be presented that two distinct etiological pathways, one primarily congenital and one primarily environmental, can culminate phenotypically as a psychopathic personality.

#### HISTORICAL QUESTS: INDICIA, ROOTS, INDICIA

Inaugural formal discussions of psychopathy were descriptive, emphasizing definitions and diagnostic "symptoms" characterizing the disorder. Early in the nineteenth century, Pinel described a group of patients exhibiting *manie sans delire* who committed inexplicable violent crimes but did not share most traits of insane patients. In 1835, Prichard (Davison & Neale, 1990) portrayed "moral imbecility" as a mental derangement in which the intellect was unimpaired but which manifested "in the state of feelings, temper or habits. In cases of this nature, the moral and active principles of the mind are strongly perverted and depraved" (p. 15). The focus on description and classification of this evil character culminated in Kraepelin's (1915) nosological system in which seven types of psychopathy were delineated (e.g., antisocial, liar/swindler, impulsive) each according to the foremost symptom, later expanded by Schneider to embody 10 types (although clearly different from Hare's [1996] and Cleckley's [1982] construct).

Aside from occasional mentions of "hereditary taint" (e.g., Krafft-Ebing, 1939), little consideration was devoted to causal hypotheses in the first century of the formal evolution of the psychopathy construct (Cleckley, 1982). This trend began to change in the late 1920s with a trickling of etiological theories being published (e.g., Partridge, 1928, 1929). Following Cleckley's classic discourse on psychopathy, *The Mask of Sanity*, in which a greater understanding of the disorder was demanded, theories emerged fast and furious from the 1940s to the 1960s from the minds of learning theorists (e.g., Hill, 1954; Mosher, 1965; Schacter & Latane, 1964), psychoanalysts (e.g., Arieti, 1963; Donnelly, 1964; Levine, 1940; Thorne, 1959), and psychophysiologists (e.g., Darling & Sandell, 1952; Hare, 1970). In a bibliogra-

phy published in 1967 by Hare and Hare, 218 studies were subsumed under a heading of etiology — more than any other category.

Despite such aggregation of research and theory on psychopathy, the causes of the disorder remained and remain nebulous. The 1970s to the present have witnessed a return to the early definitional focus resulting in Hare and his colleagues' construction of an excellent reliable tool for diagnosing psychopathy in criminal populations (Hare [PCL], 1980, [PCL-R] 1985; Hare, Harpur, Hakstian, Forth, Hart, & Newman, 1990; Hart, Hare, & Harpur, 1992). In the laboratory, many intriguing consistent correlates of psychopathy have been established in affective, semantic, and physiological domains (for reviews see Hare, 1993, 1996), some suggestive of the idea of "faulty wiring" (Hare, 1993, p. 169) — that psychopaths probably have some neurological (likely genetic) anomalies creating a predisposition to the disorder (Livesley, Lang, Jackson, & Vernon, 1992). For example, psychopaths show deficiencies in processing emotional information (e.g., Patrick, Bradley, & Lang, 1993) and seem highly adept at focussing on events of interest to them while selectively "tuning out" threatening stimuli (e.g., Ogloff & Wong, 1990). As well, the construct clearly has utilitarian value in forensic contexts (e.g., predicting parole violations; Hart, Kropp, & Hare, 1988). Clearly, with the breadth of this descriptive knowledge base, concentrated etiological venturing and testing are felicitous pursuits. It is unfortunate that possible environmental factors involved in the genesis of psychopathy have been swept under the rug. Possible reasons for this trend include the apparent absence of reliable experiential precursors, the current contagion of exciting and sophisticated laboratory research, and the prevailing "political" climate within criminological academia.

## SUGGESTIONS OF EXPERIENTIAL FACTORS

As with many of the psychopathologies, dominant thinking on the nature/nurture debate in psychopathy has shifted dramatically over time. As previously noted, a current zeitgeist within forensic psychology is to discount possible adverse childhood experiences as precursors of adult criminality. In this recognized "age of the victim" it seems exculpating to trace criminality to early experience (the "abuse excuse"). Contesting the vague notion of "hereditary taint," many earlier theorists maintained that in most cases of psychopathy, psychogenic causes could be established if adequate investigation were made (e.g., Karpman, 1947; Greenacre, 1945). This view culminated in a classic review of the literature by McCord and McCord (1964) who rendered the judgment that extreme parental rejection and lack of affection were the primary causes of psychopathy. Similar views have also been expressed by mental health practitioners; psychiatrists David Tomb and Daniel Christensen (1987) argued that "In spite of the points above [describing genetic perspectives], criminality and antisocial personality disorder are most strongly correlated with unstable, unhappy childhood environments, not biology" (p. 125).

Few longitudinal studies have looked specifically at relations between childhood experiences and adult psychopathy; instead most have examined adult criminality (e.g., see Feldman, 1977). An exception was research by Robins (1966) who conducted a seminal anterospective study examining a large sample of adults who, as children 30 years before, had been seen in a guidance clinic and a control group of adults who had resided in the same area but had not been referred. He found that inconsistent, abusive or absence of discipline by parents predicted psychopathy in adulthood. Other studies have also found that inconsistent discipline, disrupted family life (e.g., divorce), and rejecting, physically aggressive parents are predictive of adult psychopathy (McCord, 1979; van Dusen, Mednick, & Gabrielli, 1983; Wilson & Hernnstein, 1985). Other studies have not garnered support for a relationship between familial factors and a diagnosis of psychopathy. For example, DeVita, Forth, and Hare (1990), investigating the family backgrounds of psychopathic criminals and other criminals,

found no statistical evidence for differences between the two groups but "not surprisingly, most criminals came from families marked by some sort of problem" (Hare, 1993, p. 175). The earlier position that psychopathy can invariably be traced to childhood experiences has been essentially disconfirmed with a wide recognition that most psychopaths do not come from obviously dysfunctional backgrounds (e.g., Cleckley, 1982). As well, clearly most people who experience childhood mistreatments do not eventually become psychopathic or even criminal (although some serious problems do ensue in many cases).

From this brief but representative survey of the literature it is evident that, unlike the robust laboratory findings mentioned earlier, there are no early life experiences which are consistently found to be associated with a subsequent diagnosis of psychopathy. Nonetheless, let's not commit a nonsequitur and conclude that there is no relationship. If, as this paper postulates, psychopathy has a dichotomous causation the unreliability of the findings might be explained in a couple of ways:

- Background data on two (etiological) forms of psychopaths have been inadvertently aggregated, increasing the Type II error rate (i.e., there may be reliable experiential correlates for one form masked by data pooling). This would be particularly problematic if one form, more dependent on environment, represents a small proportion of the psychopaths studied.
- 2. Researchers may have selectively studied one form of psychopathy (i.e., most studies have looked at a biased sample of incarcarated, male criminal psychopaths. It is possible that one form is not well represented in this population).

#### AN INTERACTIVE ETIOLOGICAL THEORY OF PSYCHOPATHY

## The Prevailing Perspective

Most modern theorists agree that the current notion of psychopathy likely represents "a complex — and poorly understood — interplay between biological factors and social forces" (Hare, 1993, p. 174). The enigmatic nature of this interaction reflects the unsolved mystery of: exactly what are the biological and social forces? Although not a popular view in the present societal context, there is almost certainly a considerable genetic component in the proclivity to criminal behavior by psychopaths (see Moffitt & Mednick, 1988 for a review). It is beyond the scope of this paper to discuss in depth either the possible genetic or physiological mechanisms promoting a "predisposition" to psychopathy. It is, however, necessary to comment on the presumed general nature of such propensity. Given that a critical feature of psychopathy seems to be an inability to form bonds with others (and a corresponding lack of empathy and conscience), this may represent a phenotypic manifestation of the predisposing factor(s) (Hare, personal communication, March 7, 1995). According to this "bad seed" perspective, Factor 1 characteristics are born within certain people and Factor 2 traits emerge from this predisposing "seed" (probably polygenic in nature) regardless of (although not oblivious to) environmental factors. What environmental factors will influence is the developmental path (i.e., whether the person becomes a psychopathic criminal or a psychopathic politician). For example, Ted Bundy ("the" prototypical psychopath) grew up in a generally happy, supportive family which facilitated his role as a promising law student before embarking on a series of premeditated murders. He realized in adolescence that he had no conscience and "didn't know what underlay social interactions" which he retrospectively attributed to "something that was programmed by some sort of genetic thing" (Flaherty, 1992, p. 15). A prevailing extant view, then, holds that psychopathy is a unitary construct deriving from a complex but relatively homogeneous pattern of congenital factors (precluding conscience formation) and shaped by environmental factors.

#### A "Convergent-Paths" Perspective

Speaking generally, punishment hardens and numbs, it produces concentration, it sharpens the consciousness of alienation, it strengthens the power of resistance. (Nietzsche)

As it originated in German psychiatry, psychopathic personality literally referred to a "psychologically damaged" person but has come to connote a "socially damaging person" (Blackburn, 1992). The present perspective underscores the idea that psychopathy is not a unitary construct etiologically; one type is hypothesized to result primarily from early "psychological damage" in contrast to the chiefly congenital type. Not only is etiology of psychopathy viewed as "interactive" (predisposition/environment) but also "dual" (two convergent causal pathways). Specifically, this paper proposes that there may be two types of psychopathy — the classic or "fundamental" psychopath as recognized in the prevailing view described above and a "secondary" class of psychopath — phenotypically (and diagnostically) indistinguishable in most respects. Distinct causal factors lead to a common outcome: a reduced capability or an inability to experience emotion.

There have been other proposals of a secondary type of psychopath some referring to APD (Blackburn, 1975), "neurotic psychopaths" (Maher, 1966), a sociobiological formulation (Mealey, 1995), or "acquired sociopathy" due to organic damage (Damasio, Tranel, & Damasio, 1987). But to my knowledge, none has proposed the current etiological formulation.

The present hypothesis holds that whereas the predominant form of psychopathy — fundamental (FPy) — is inevitable due to a polygenic predisposition disallowing the formation of affective bonds (although its effects may be malleable), the genesis of another form of psychopathy — secondary (SPy) — is more heavily or exclusively dependent on social forces. As well, SPy may have a different diathesis than FPy and show subtle distinctive manifestations. Unlike the fundamental psychopath (FP), born without the capacity to form the affective element typical of most humans, the secondary psychopath (SP) is hypothesized to have experienced a "de-activation" or dissociation of a developing basic affective nature and conscience (see Figure 1). The diathesis would promote a capability for detachment of one's emotions rather than a disability to experience emotion. Although oversimplified, the genotype–environment correlation might be considered "reactive" and "active" (parents act abusively toward a psychopathic child because of the child's character/child seeks "affectionless" lifestyle) in FPy and primarily "passive" (abuse occurring independent of the child's innate characteristics resulting in alexythymia) in SPy.

Such de-activation represents a coping mechanism in response to traumatic and disillusioning interpersonal experiences. In contrast to the case of FPy, (using Hare's PCL-R terminology) SPy Factor 1 traits (interpersonal/affective symptoms) develop during childhood with Factor 2 features (socially deviant behaviors) emerging later in childhood and adolescence according to the progressive deterioration of emotional experience. As will be elucidated shortly, it is argued that SPy, with its de-activation of emotion, might be considered a dissociative disorder rather than a personality disorder.

## THE PROPOSED DISSOCIATIVE MECHANISM

As with all dissociative phenomena (Spiegel & Cardena, 1991), the processes involved here are abstruse. Proceeding on a speculative level, in the SP there is a capacity for empathetic responding but it is "turned off" with repeated disillusionment of the child through physical or sexual abuse or other mistreatment (see Figure 1). This should be considered a dissociative disorder (see Spiegel & Cardena [1991] for formal criteria) with the child's emotion being dissociated from or unconnected with cognition and behavior over time. Eventually, as an efficacious distress reduction strategy, affect becomes inactivated or even abrogated. The strategy of "not feeling" becomes reinforced, with reduced psychological distress or trauma

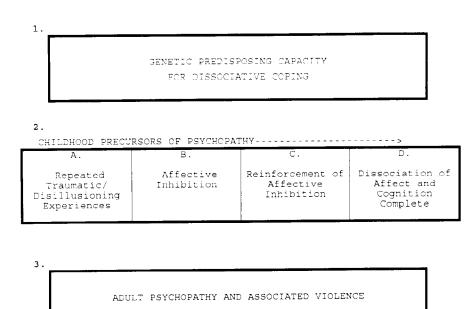


FIGURE 1. Etiological Pathway to Secondary Psychopathy.

associated with abusive incidents. Re-experiencing abusive events in memories would also be made less painful or disillusioning in absence of affect serving as further positive reinforcement for maintenance of the coping technique. As Cicero observed: "There is something pleasurable in calm remembrance of a past sorrow."

Unfortunately, few studies have examined prospectively long-term alterations in emotional functioning following abusive incidents in childhood. Studies generally find an increased level of aggressiveness in abused children (Wilson & Hernnstein, 1985) and a few have mentioned that many abused children become listless, excessively vigilant (an interesting parallel with the psychopath; Ogloff & Wong, 1990), and totally unresponsive emotionally (Aber & Cicchetti, 1983). Vaillant and Perry (1985) argued that some psychopaths traumatized during childhood as the result of parental separation were *guilt-ridden* and disillusioned during and after separation, indicating an earlier capacity for emotional experience.

There is evidence that traumatic experiences can have profound qualitative effects on affective functioning (Davidson & Foa, 1991), first described by Pierre Janet as the psychological process in which "the organism reacts to overwhelming psychological trauma" (cited in van der Kolk & van der Hart, 1989). One of the key diagnostic symptoms of Posttraumatic Stress Disorder (PTSD) is "persistent avoidance of stimuli associated with the trauma or numbing of general responsiveness" (although note that PTSD is currently classified as an anxiety disorder rather than a dissociative disorder). Evidence for presence of this symptom includes efforts to avoid thoughts or feelings associated with the trauma, feeling of detachment or estrangement from others, and restricted range of affect (DSM-IV, 1994). Kingsbury (1988) notes that emotional numbing, emotion suppression, and avoidance may occur in response to repetitive negative emotional experiences. Wilkinson (1983) surveyed survivors of the Hyatt Regency Skywalk collapse in which 114 people died. An astounding 36% of respondents reported no longer being capable of feeling deeply about anything. Similarly, Valent (1984) found that there was a common absence of emotions in survivors of a natural disaster.

Studies looking at PTSD in children have indicated that PTSD criteria apply to children as well as adults (Pynoos et al., 1987). In the case of child abuse, some child abuse victims show

a profound affective blunting. Everstine and Everstine (1989) observed that many severely abused children learn to diminish the emotional impact of disillusionment by "turning off" their emotions, which is rewarded through the reduction of negative experience. As adults, the result of this well-developed dissociative reaction is the presentation of a "hardened" individual with a "strong/tough demeanor" (p. 156). It seems plausible that, in certain cases, such emotional deactivation could contribute to creation of a psychopath. Depending on age of abuse onset, the affective component and "conscience" of the child might be in an early, late, or complete stage of development which might influence the level of resistance to a dissociative strategy.

In psychodynamic terms, absence of guilt, empathy, and other emotional traits in psychopaths is founded in faulty superego development. In Civilization and Its Discontents (1961), Freud argued that conscience formation is dependent upon a child's wish to exact vengeance for the authority and control exerted by the parents. Direct revenge is not possible because the child needs the love of the parent; instead the child masters the situation by identifying with the controlling parent and treating the id impulses as the parent treated him/her — by controlling them. Obedience to authority becomes internalized in this process referred to by Loevinger (1966) as "mastery through reversal of voice" or mastery gained by repeating what one has experienced passively. If any stage of this conscience formation is interrupted, fixation may result and continue into adulthood. For example, in the case of parental rejection, mistreatment, or traumatization (which precludes identification), fixation will occur at an early stage of conscience formation (Fenichel, 1945). As an adult, id impulses will prevail because of the ineffectual infirm or incomplete superego. Indeed, in most formulations of conscience origination the perception of parental love is cardinal (e.g., Hill, 1960; Maher, 1966); the disillusionment of recognizing no parental love is presumably ruinous for normal affective development.

The present hypothesis states that psychopathy can be acquired through learning to deal with painful disillusioning stimuli (e.g., abuse). Harpur and Hare (1990) reviewed evidence that psychopaths make effective use of active coping strategies to deal with aversive stimulation. For example, noting that psychopaths show anticipatory heart rate acceleration and small electrodermal responses to aversive stimuli, Hare (1978) suggested that they show an active coping response allowing them to tune out premonitory cues. As well, psychopaths are proficient at allocating their attentional resources to less aversive stimuli in the environment (e.g., Hare, 1982). It is plausible that this coping strategy could develop in certain individuals who are traumatized repeatedly in childhood.

Studies looking at lexical decision-making have indicated that nonpsychopaths recognize emotional words faster than neutral words, whereas psychopaths do not show such a difference reminiscent of Cleckley's (1976) referral to "semantic aphasia." Interestingly, Williamson, Harpur, and Hare (1991) unexpectedly found that psychopaths actually took longer (although not statistically significant) to recognize emotional words (especially negative) than neutral words. The longer mean time was attributed to one psychopathic subject and was viewed as a "perceptual defense" against emotional stimuli prompting the authors to suggest that future research consider whether psychopaths show a reliable interference effect when processing emotional material. It is possible that this subject was an SP engaging in an emotional muting strategy learned in childhood in the avoidance of psychological distress. It would be interesting to investigate the background of this subject to see if it is consistent with the present formulation.

## RESEARCH POSSIBILITIES AND IMPLICATIONS

The dual-etiological hypothesis is speculative and somewhat difficult to test. The best (and arguably the only) method to assess the validity of the "secondary psychopath" idea is with a longitudinal, prospective design examining a large sample of children known to have been physically or sexually abused by a parent (i.e., conviction) and a matched control sample

(similar to the Robins [1966] strategy), excluding children known to have suffered any form of head injury as a result of violence. Next, a thorough investigation would be conducted looking at preabuse (baseline) affective functioning and emotional patterns concurrent with and subsequent to the time of the abusive incidents. The nonabusing parent (if there is one), siblings, relatives, family friends, teachers, etc. would be interviewed for evidence of emotional blunting in the children. Of greatest interest would be cases in which strong evidence indicates capacity for emotional experience prior to abuse onset followed by progressive deterioration of affect over time (Group 1). This population of children would be followed up longitudinally and compared to abused children who displayed normal emotion prior to abuse and did not show evidence of emotional blunting after abuse onset (Group 2). Cases in which there is evidence of little emotion or empathy prior to abuse onset would be identified (i.e., these children might be FPs — Group 3). The final population would be the matched group of nonabused control children (Group 4). In adulthood, all individuals (ideally) would be assessed for psychopathy by interviewing the individuals themselves, if possible, as well as their family members and associates. As well, the possibility that SPs and FPs differ in their clinical manifestations would be addressed by examining possible differences in the frequency of individual PCL-R criteria for the presumed FPs and SPs.

The major predictions of the "secondary psychopath" model include: (a) a high proportion of Group 1 subjects would be diagnosed as psychopaths in adulthood (SPs); (b) no Group 2 subjects would be diagnosed as psychopaths (having already displayed normal affective development in childhood); (c) the highest proportion of psychopaths would emerge in Group 3, children who from the beginning showed a poverty of affect (FPs); (d) a very small proportion of Group 4 subjects would be diagnosed as psychopaths (approximately equalling the prevalence rate of FPy in the general population).

As mentioned earlier, one possibility is that SPs and FPs would differ subtly in their clinical manifestations. Clearly, people diagnosed as psychopaths with Hare's Psychopathy Checklist (scoring at least 30/40) exhibit widely variable patterns of behavior, largely because of the polythetic nature of the construct. Some qualities may coincide more with the polygenic predisposition than simply resulting from lack of affect. If this is the case, SPs would be less likely to exhibit such traits. For example, some psychopaths do not exhibit the manipulativeness and "user quality" usually associated with the disorder (Hare, personal communication, March 7, 1995). Cleckley (1982) devoted an entire chapter in his text to "incomplete manifestations of the disorder," describing individuals who, despite a poverty of affect and fitting most criteria for the disorder, did not generally manipulate or harm others. On the other hand, it is tenable that all diagnostic features of psychopathy are directly related to absence of affect, regardless of origin, so there might not be any distinguishing diagnostic traits between FPy and SPy. In this case, the absence of certain traits in some psychopaths would have to explained in another way.

#### **CONCLUDING REMARKS**

As I was concluding this paper, a disturbing report appeared in the *Vancouver Sun* with the headline "Children appeared emotionally dead to foster parents after ordeal" (March 25, 1995, pp. A1–2). The story focused on a family who had taken as foster children three victims of one of Canada's worst cases (sixty-five alleged perpetrators) of child sexual abuse. Over the next 5 years, the new parents observed conduct problems in the children but "Perhaps most alarming, the children seemed emotionally dead. They never cried, they appeared to have no feelings . . It was as if they were operating on automatic pilot." After years in a loving environment they improved substantially, regaining some capacity for affective experience. But, without intervention, would one or all of these children have developed psychopathic personalities?

To this point, no definitive insight has been gained for the causes of any of the personality disorders (Sue, Sue, & Sue, 1990). As with most theoretical formulations in the area of personality, the hypothesis presented here is admittedly speculative; it is extremely difficult to establish causation for adult disorders from particular childhood experiences. Nonetheless, it is crucial that theories be developed and tested particularly in the case of the psychopath, a character capable of imparting inestimable harm on society. Obviously, there are problems with the proposed hypothesis. A salient example is the fact that many more females are (sexually) abused than males but there may be a higher proportion of male psychopaths in the general population. One possibility is that hysteria and/or MPD, which are seen much more commonly in females, and psychopathy are separate manifestations of the same etiological process (Cloninger, Reich, & Guze, 1975). Another weakness is that it is impossible to prove and must rely solely on correlational data for supportive evidence.

This article suggests the possibility that over time negative environmental experiences can sometimes contribute to deactivation or vitiation of normal human emotion and eventually lead to psychopathy. The present view should not be taken as a "bleeding heart" argument for exculpation or exoneration for the ills bestowed upon others by psychopaths. Despite absence of empathy for others, the *volition* of secondary (and fundamental) psychopaths is presumably perfectly functional. If the secondary psychopathy category receives continued validation, the salient implications relate to intervention. These individuals might represent a population for which early intervention or treatment in adulthood might be beneficial for society (despite the likelihood that psychological intervention almost certainly won't work with psychopaths in general; e.g., Hemphill, 1991).

The propensity of psychopaths to commit violent crimes is well-documented (c.g., Hare & McPherson, 1984; Wong, 1984). The ultimate goal of this etiological theory of psychopathy is not to amend psychopaths, but rather to reduce the violence and corruption they effects. Until the causal foundation(s) for psychopathy has been established, we should not discount the influence of either nature or nurture. In the provident words of Cleckley (1982), "let us admit the incompleteness of our knowledge and modestly pursue our inquiry" (p. 261).

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